Resistant Mycoplasma Pneumonia

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ABSTRACT

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Mycoplasma pneumoniae is recognized as a leading cause of community-acquired lower respiratory tract infection in children, which accounts for about 25-30% of pneumonia in children. Macrolides are the first-line treatment for M. pneumoniae infections. However, the extensive use of macrolides in clinical practice has resulted in the emergence of macrolide-resistant M. pneumoniae (MRMP), which has a negative impact on treatment outcomes.[1]

Here we present a case of severe, Macrolide-resistant Mycoplasma pneumoniae infection in a previously healthy 9-year-old male, culminating in necrotizing pneumonia, complicated by bilateral pleural effusion. The case highlights the challenges in diagnosing and managing atypical pneumonia unresponsive to first-line therapies.

Keywords: Pneumonia, Mycoplasma pneumoniae, Resistant Mycoplasma pneumonia, Pleural effusion, Biofire, Mycoplasma IgM.

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CLINICAL PRESENTATION

A 9-year-old male was brought to the outpatient department with a 7-day history of fever. Initially low-grade and intermittent, the fever progressed to a continuous high-grade pattern. Associated symptoms were nocturnal, dry cough persisting for 5 days from the onset of fever, and four episodes of watery, foul-smelling, non-bloody diarrhea over two days.

Despite receiving oral antibiotics and antipyretics, the patient's fever persisted, prompting hospitalization at a local healthcare facility on day 5 of illness. Initial investigations revealed leukocytosis and elevated C-reactive protein (CRP). Empirical therapy with intravenous Ceftriaxone and oral Azithromycin was initiated. However, fever and respiratory symptoms remained unchanged. Follow-up investigations ruled out dengue and enteric fever, while chest Xray demonstrated right lower lobe consolidation with parapneumonic effusion. Antibiotics were escalated to intravenous Piperacillin-tazobactam and oral Clarithromycin.

On day 7 of illness, the child had persistent high-grade fever (peaking at 105°F), tachypnea, and oxygen desaturation in room air. So the patient was referred to our tertiary care center for further evaluation and management.

HOSPITAL COURSE:

Upon admission to the pediatric intensive care unit (PICU), the patient was febrile and tachypneic requiring supplemental oxygen at 3 L/min via mask. Auscultation revealed diminished breath sounds over the right infra-axillary, inframammary, interscapular, and infrascapular lung fields with bronchial breathing over the same areas.

Laboratory investigations revealed:
Total leukocyte count: 10,550/mm^3 (N94 L5 M1)
CRP: 11 mg/dL
ESR: 28 mm/hr
Haemoglobin: 12.1 g/dL
Serum sodium: 135 mEq/L
Potassium: 3.1 mEq/L

Chest radiography showed lobar consolidation over the right side with bilateral pleural effusion **(Figure 1).** Thoracic ultrasonography identified bilateral pleural effusions, more pronounced on the right.

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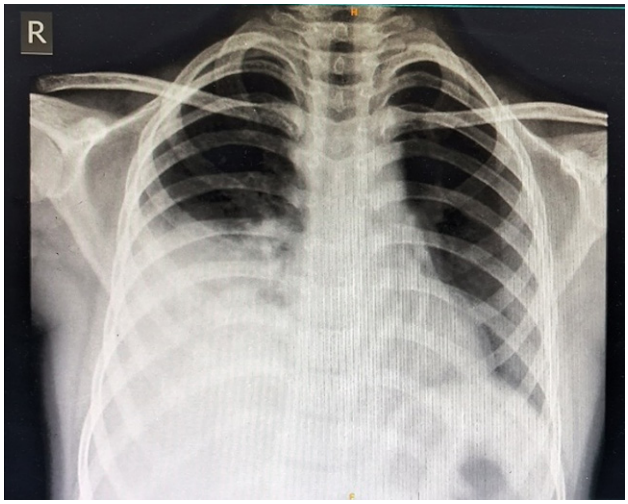

Figure 1. **Chest xray taken on day 1 of admission**

The patient was started on intravenous Piperacillin-tazobactam, Clindamycin, and oral Clarithromycin.

On day 2, 400 mL of straw coloured pleural fluid was aspirated under ultrasound guidance. Analysis of the fluid revealed:

Albumin: 2.5 g/dL

Glucose: 152 mg/dL

Total cells: 280/mm³ (98% lymphocytes)

Protein: 3.0 g/dL

LDH: 696 IU/L

RBCs: 10–15/hpf

Because of persistent fever and pleural effusion, tuberculosis workup was done which came back negative. A multiplex respiratory pathogen panel (BioFire) was positive for Mycoplasma pneumoniae. Mycoplasma IgM and IgG were also positive. Pleural fluid culture and blood culture reports were negative.

Computed tomography (CT) of the chest **(Figure 2)** demonstrated mild to moderate right pleural effusion extending into the right major fissure and upper paramediastinal region. Additional findings included mild left-sided effusion, passive subsegmental atelectasis of the left lower lobe, and complete consolidation of the right lower lobe with signs suggestive of early necrotizing changes. The right upper and middle lobes showed subsegmental collapse-consolidation.

In view of poor clinical response and imaging findings, Clindamycin and Clarithromycin were discontinued on day 4 of admission. The patient was commenced on intravenous linezolid. Suspecting MRMP patient was started on oral Doxycycline. Supportive measures included nebulization, chest physiotherapy, oxygen supplementation, and antipyretics.

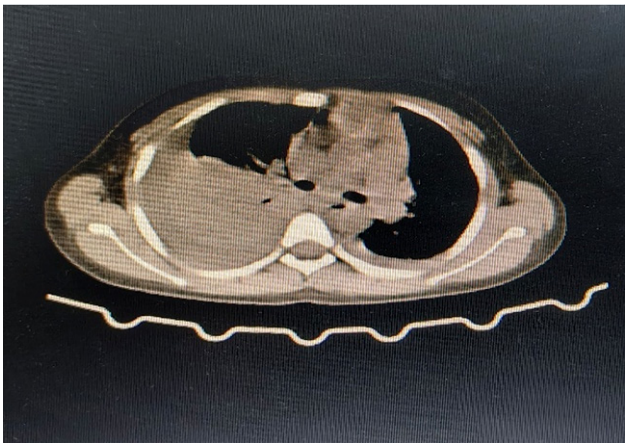

Figure 2. **CT chest showing mild to moderate right pleural effusion extending into the right major fissure and upper paramediastinal region**

Due to ongoing symptoms, a second ultrasound-guided pleural tap was done on day 6, yielding 350 mL of straw-colored fluid. Repeat fluid analysis revealed:

Albumin: 2.5 g/dL

Glucose: 111 mg/dL

Total cells: 650/mm³ (91% lymphocytes)

Protein: 3.5 g/dL

RBCs: 2–4/hpf

On day 7 antibiotic therapy was further escalated to intravenous Meropenem. Clinical improvement followed, with better oxygenation, improved breath sounds over the right Inframammary, infraaxillary areas and gradual subsidence of fever. However, intermittent fever persisted. Since patient was not responding to Doxycycline, intravenous Levofloxacin was initiated on day 10. Fever subsided by day 12. Air entry improved over the right interscapular and infrascapular areas, and bronchial breath sounds were auscultated over the right

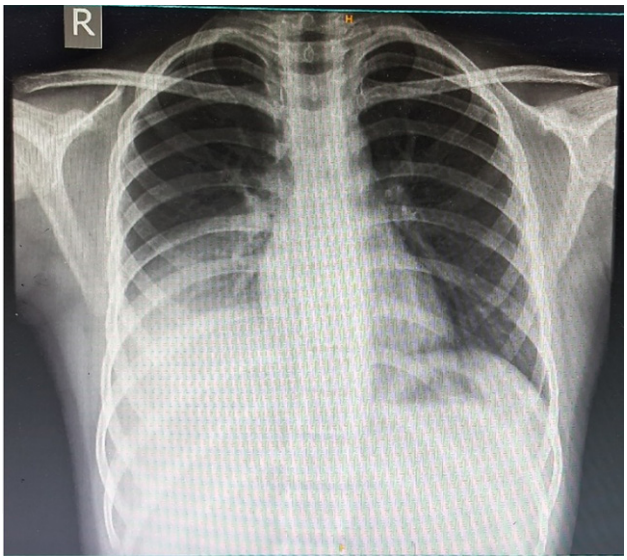

Figure 3. Repeat chest Xray showing resolving pneumonia

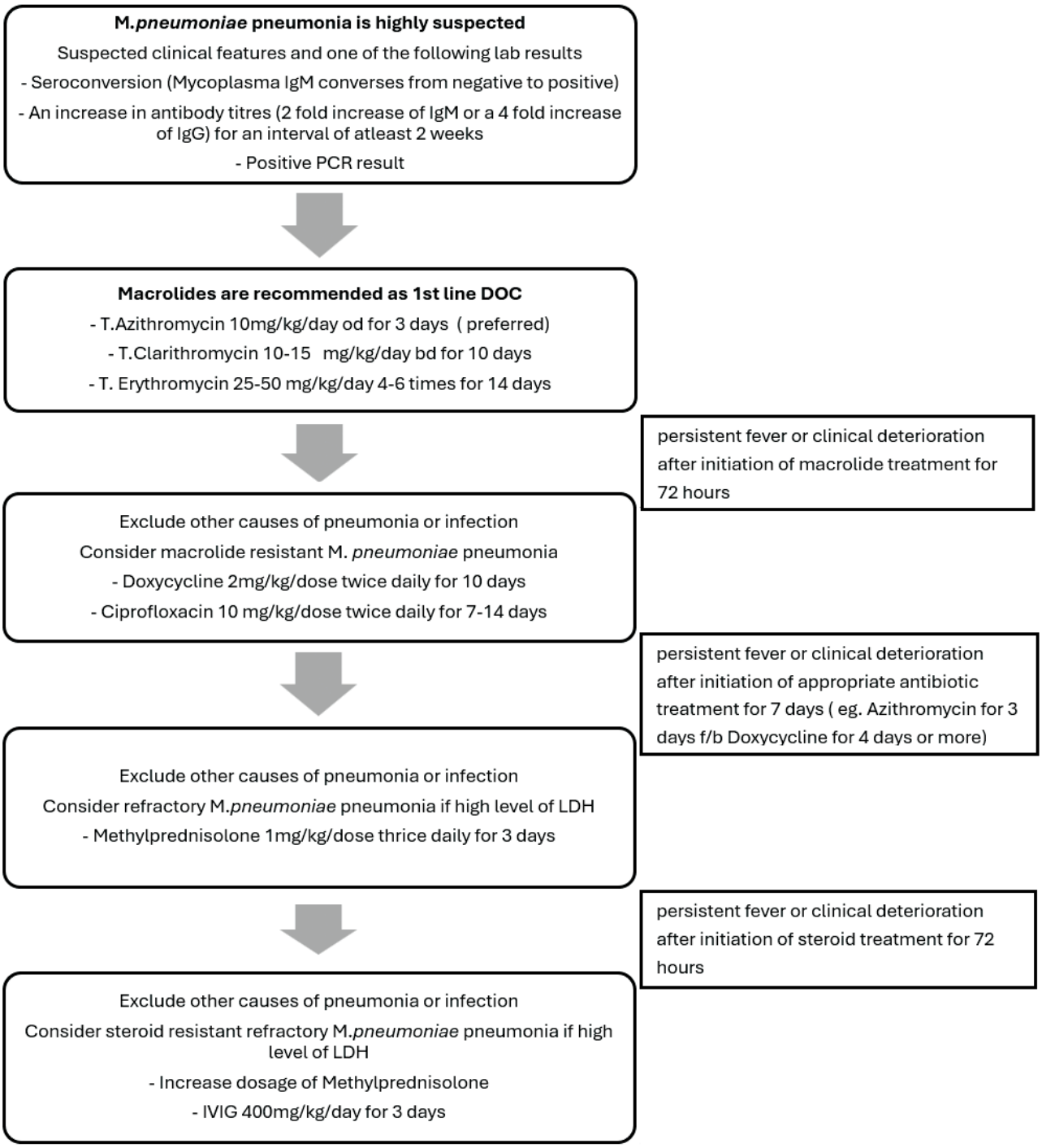

Figure 4. **Rational stepwise approach for treating Mycoplasma pneumonia in children**

interscapular area. Parenteral antibiotics were changed to oral Levofloxacin and Faropenem on day 13. Repeat chest Xray showed resolving pneumonia with parapneumonic effusion **(Figure 3).**

The patient's clinical condition steadily improved. He was discharged in a stable state on day 16 with the advise to complete a 14-day course of oral Levofloxacin and Faropenem.

CONCLUSION

Mycoplasma pneumoniae is a common causative pathogen in community-acquired pneumonia (CAP) during childhood. In the post–pneumococcal conjugate vaccine (PCV) 13 era, the epidemiology of pediatric pneumonia has changed. In some countries where PCV13 is already included in national immuniza-

tion program, M. pneumoniae has become the leading pathogen in pediatric CAP.[2,3]

Previously Mycoplasma pneumonia had been described as "walking pneumonia" due to its mild and self limiting clinical presentation. However, life-threatening pneumonia or even acute respiratory distress syndrome requiring extracorporeal membrane oxygen has been reported.[4] Furthermore, some extrapulmonary symptoms, such as mucositis, hepatitis, encephalitis, autoimmune hemolytic anemia, or erythema multiforme, have linked M. pneumoniae infection to the formation of autoimmunity or immune complexes. The association between M. pneumoniae and refractory asthma has also been mentioned.[5]

Diagnosis is complex, as clinical symptoms alone are insufficient; key indicators include prolonged fever, intractable or pertussoid cough, elevated CRP with normal WBC, and poor response to beta-lactams. Diagnostic methods include cold agglutinin testing (limited specificity), serology (IgM and paired IgG), and PCR, with the latter considered the gold standard. Treatment is complicated by the organim's lack of a cell wall, making it resistant to beta-lactams. Macrolides, particularly Azithromycin and Clarithromycin, are the first-line agents due to both antimicrobial and anti-inflammatory properties. However, macrolide-resistant M. pneumoniae (MRMP), attributed to mutations like A2063G in the 23S rRNA gene, is an emerging issue, with resistance rates varying globally, up to 90% in China and significantly high rates of resistance is also seen in other Asian countries including Taiwan, Japan, South Korea etc. Tetracyclines like Doxycycline and fluoroquinolones like Levofloxacin are used as second-line agents, especially in macrolide failures. There is no definitive guideline for steroid use, but they are considered in severe pulmonary disease and extrapulmonary complications. Given below is a flowchart depicting the rational stepwise approach for treating Mycoplasma Pneumonia in children.[6]

END NOTE

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